

CASE REPORTS

VENTRICULAR FIBRILLATION IN MYXŒDEMA HEART DISEASE WITH SPONTANEOUS REVERSION

BY

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Transient ventricular fibrillation with spontaneous reversion to sinus rhythm has been recorded many times in cases of heart block with the production of Stokes-Adams attacks (Schwartz and Jezer, 1932; Parkinson, Papp, and Evans, 1941). The duration of the ventricular fibrillation in these cases has varied from a few seconds to just over six minutes in two cases.

These cases excepted, spontaneous reversion is very rare. The case reported here is one in which it occurred after three minutes in a patient with myxœdema heart disease and recent myocardial infarction.

Case Report

Mrs. A. B., aged 72, was admitted to the Central Middlesex Hospital at midnight on October 23, 1962. Two hours previously she had developed severe retrosternal pain which had persisted. On admission she was sweating and cyanosed but the pulse rate and blood pressure were normal. The heart sounds were also normal. The jugular venous pressure was raised 4 cm. above the sternal angle and there was pitting œdema of the ankles. The electrocardiogram showed the patterns of extensive anterior myocardial infarction (Fig. 1). She was given pethidine 100 mg. with levallorphan tartrate 1.25 mg. by intramuscular injection.

She had improved by the following morning when her myxœdematous facies was recognized, and the classical symptoms and signs of this condition were elicited. The serum glutamic-oxalacetic transaminase was 210 units/ml. and the cholesterol 400 mg./100 ml.

The next morning while a further electrocardiogram was being recorded she collapsed. The tracing showed a run of ventricular ectopic beats at the rate of 180 a minute, succeeded by exactly three minutes of ventricular fibrillation (Fig. 1). During the period of ventricular fibrillation no pulse could be felt and no heart sounds were audible. The patient looked dead. The sternum was depressed vigorously six times during this period. Mouth-to-mouth breathing was not attempted. Spontaneous reversion to sinus rhythm occurred about one minute after the end of this procedure and further electrocardiograms showed a transient pattern of injury.

The immediate recovery was complicated by an attack of pulmonary œdema which was treated with digoxin 1 mg. and aminophylline 0.25 g. intravenously. One hour later she had a generalized convulsion and was given 5 ml. paraldehyde by intramuscular injection. No further fits occurred.

She recovered consciousness after 18 hours, and 72 hours later she appeared to have recovered completely. There was no evidence of neurological defect. Treatment continued with oral digoxin and anticoagulants, and apart from one episode of mild anginal pain recovery thereafter was uncomplicated.

Thyroid function tests confirmed the clinical diagnosis of myxœdema. A radioactive-iodine urinary excretion test showed reduced thyroid function, the "T" index being 2.2 (Fraser *et al.*, 1953). The uptake of ¹³¹I-tri-iodothyronine by the red cells was 12.6 per cent, the normal range with this method being 15–20 per cent (Goolden *et al.*, 1962).

Chest radiograph revealed cardiac enlargement due to left ventricular dilatation. There was pulmonary venous hypertension, and bilateral pleural effusions were present. Radioscopy showed poor cardiac pulsation.

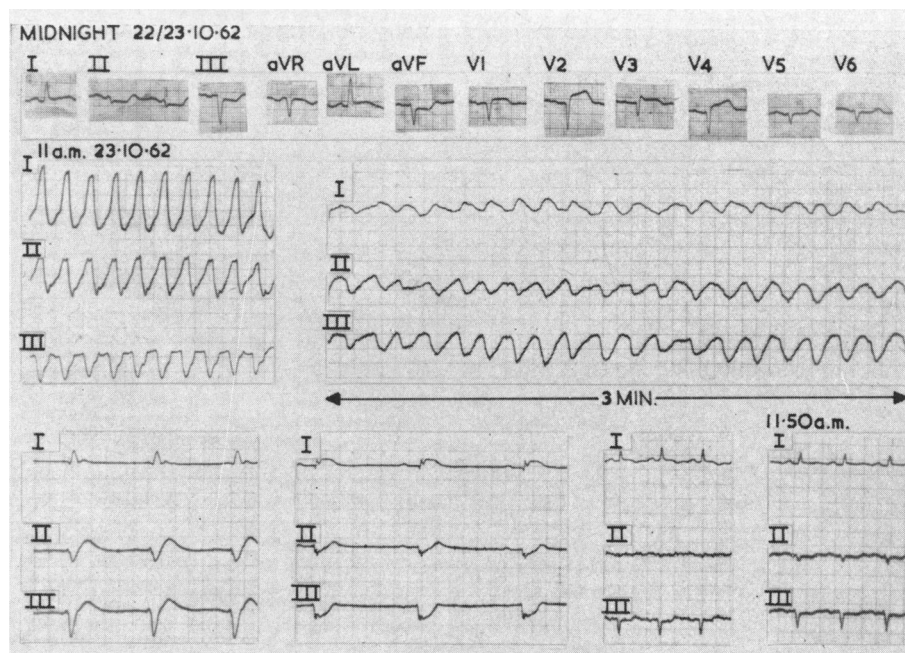


FIG. 1.—Electrocardiograms showing (top) standard limb and chest leads on admission; (middle left) standard limb leads during ventricular tachycardia, and (middle right) during ventricular fibrillation; (bottom) successive tracings after reversion to sinus rhythm.

Six weeks after admission treatment with L-thyroxine 0.05 mg. daily was started. Four days after starting thyroxine she suddenly developed a further attack of pulmonary oedema and died.

At necropsy the heart weighed 410 g. There was a moderate-sized straw-coloured pericardial effusion. The anterior, apical, and antero-septal walls of the left ventricle were destroyed by a recent organizing infarct and there was marked atheromatous narrowing of the left descending coronary artery, with a recanalizing thrombotic occlusion in its third centimetre. There was no evidence of fresh infarction.

The thyroid gland weighed 8 g. and on microscopy the typical appearances of Hashimoto's thyroiditis were seen.

Discussion

Apart from Stokes-Adams attacks, transient ventricular fibrillation with spontaneous reversion has been recorded in ischaemic heart disease (Priest, 1949; Choquette *et al.*, 1956; Semple and Dall, 1962; Harden, Mackenzie, and Ledingham, 1963), in rheumatic heart disease (Sampson quoted by Sokolow and Ball, 1956), in the course of cardiac catheterization in a patient with a normal heart (Gordh, Linderholm, and Ström, 1956), in acute or chronic renal failure (Wetherill and Nixon, 1962), and in the absence of organic heart disease (Dock, 1929; Moe, 1949; Storstein, 1949; Stern, 1957). The present case is, therefore, the twelfth reported case of spontaneous reversion from ventricular fibrillation without previous heart block.

It is commonly believed that full oxygenation of the myocardium is essential before spontaneous or electrical defibrillation can occur. However, in this patient neither external cardiac massage nor mouth-to-mouth breathing was undertaken during a period of ventricular fibrillation known to have lasted three minutes. It must be assumed that for this period there was failure of both coronary and cerebral circulations. It may be that the heart and brain were better able to stand this period of anoxia because of the reduced demands for oxygen by the tissues in myxoedema.

Summary

Spontaneous reversion from ventricular fibrillation to sinus rhythm in a 72-year-old lady with myxœdema heart disease recently complicated by myocardial infarction is reported.

A continuous electrocardiographic record showed the episode to be of exactly three minutes' duration. The rarity of spontaneous reversion is brought out by a review of previously reported cases. The relation of anoxia to this is discussed.

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